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HYPERTROPHY OF THE MUSCULAR WALLS OF THE MINUTE ARTERIES IN CASES OF CHRONIC BRIGHT'S DISEASE.*

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IN order to render intelligible the specimens which I am about to show you, I must refer to some well known anatomical facts and physiological doctrines.

The larger arteries, both pulmonary and systemic, have their walls mainly composed of yellow elastic tissue with but a small proportion of muscular tissue. The minute microscopic arteries, bordering on the capillaries, have their middle coat entirely composed of muscular tissue, the nuclei and fibres encircling the canal of the artery. The arterial trunks and their larger branches, by the elastic resiliency of their walls, convert the intermitting jet of blood from the heart into a continuous stream in the minute arteries and capillaries. The elasticity of the larger arteries acts as a propelling force in aid of the heart. The distending force of the ventricle is partly communicated to the walls of the arteries, where it is stored up as mechanical force, and it is imparted to the column of blood by the elastic resiliency of the arterial walls during the diastole of the ventricle. The whole of the propelling force which ordinarily acts upon the arterial blood is derived from the muscular contraction of the ventricle. We need not now take into consideration the influence of the respiratory movements upon the circulation. The muscular force of the heart acts upon the blood, directly during the systole of the ventricle, indirectly during the diastole, through the elastic resiliency of the arterial walls. The elastic force of the arterial walls is as obviously derived from the muscular power of the heart as the elastic force of an archer's bow is derived from the muscles which bend the bow. The minute arteries with their muscular walls have

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an entirely different function from the larger arteries. The minute arteries, under the influence of the vaso-motor nerves, regulate the blood-supply to the various organs and tissues. By their contraction the canals are narrowed, and the blood-stream is in a corresponding degree diminished; on the contrary, their relaxation enlarges the arterial canals, and a fuller stream of blood is permitted to pass. The minute muscular arteries perform the functions of stop-cocks. They do not drive on the blood by their contraction; on the contrary, the active contraction of the minute arteries is antagonistic to the active contraction of the ventricle. The narrowing of the arterial canals opposes an obstacle to the onward movement of the blood, and the heart then has to beat with increased force in order to carry on the circulation.

More than thirty years ago (in the 1st volume of *Guy's Hospital Reports*), Dr. Bright pointed out the fact that in a large proportion of cases of chronic Bright's Disease there is marked hypertrophy of the left ventricle of the heart, even when the valves and large arteries are free from disease; and he suggested, as a probable explanation of this, that "the altered quality of blood might so affect the minute and capillary circulation as to render greater action necessary to force the blood through the distant subdivisions of the vascular system."

Twenty years ago, in the 33rd volume of the *Medico-Chirurgical Transactions*, I described and figured hypertrophy of the muscular walls of the minute renal arteries as occurring almost constantly in cases of chronic Bright's Disease. At that time the function of the minute muscular arteries was more doubtful than it now is, and I erroneously supposed and suggested that this hypertrophy of the muscular walls of the minute renal arteries was the result of an effort on their part to help on the blood through the compressed and obstructed vessels in front.

Since the publication of that paper, physiological experiment and pathological research have thrown much light upon the influence of the small arteries upon the circulation both in the healthy and in the diseased states of the system.

About three years ago, it occurred to me that the hypertrophy of the left ventricle in cases of chronic Bright's disease might be a result of increased contraction of the small arteries, excited by the abnormal quality of the circulating blood; and I went on to argue that, if this were so, we should find evidence of the fact in the existence of hypertrophy of the muscular walls of the minute arteries in various tissues. The next step was to search for this hypertrophy in cases of chronic Bright's disease associated with hypertrophy of the left ventricle. Accordingly, we sought for it, and found it unmistakably present, not

only in the arteries of the kidney, but also in those of the pia mater, the skin, the intestines, and the muscles. It probably exists in the arteries of other tissues which we have not examined.

I have published an account of these observations in the fifty-first volume of the *Medico-Chirurgical Transactions*.

We have found this hypertrophy of the minute arteries in various tissues associated with hypertrophy of the left ventricle in every case that we have examined, amounting now to more than a dozen. In fact, it occurs with the constancy of a physical law. In some cases, the arterial hypertrophy appears to be about equal in all the tissues, the kidney excepted; while in other cases it seems to be greater in some tissues than in others. The amount of hypertrophy is ascertained by a comparison with normal arteries of the same size.

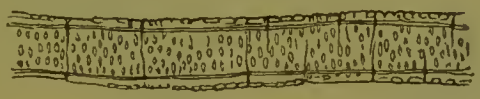

Fig. 1.—Normal artery from the kidney, showing the thickness of the walls in proportion to the canal. In the muscular wall there is an outer circular layer and a thin inner longitudinal layer.—X 200.

The arteries are more readily discovered when filled by a coloured injection, such as Prussian blue; and I am much indebted to my friend and colleague Dr. Kelly for the labour that he has bestowed on the investigation of this subject and the preparation of specimens.

There seems to be a direct relation between the hypertrophy of the left ventricle and that of the minute arteries in the various tissues; and in many specimens the walls of the arteries may be seen to have at least three times their normal thickness.

The minute *renal* arteries are usually more hypertrophied than those from other tissues; and the hypertrophied renal arteries present the peculiarity of having an inner longitudinal layer of fibres equal in thickness to the outer circular layer. (See Fig. 2.)

Fig. 2.—Artery with hypertrophied walls from the kidney. The inner longitudinal layer of fibres is equal in thickness to the outer circular layer. The canal of the vessel is filled with injection.—X 200.

This inner longitudinal layer of fibres is only just visible in the arteries from other tissues.

The thickening of the arterial walls is an undoubted instance of genuine hypertrophy. It is an increased growth of a normal tissue, without change of texture. The walls of the hypertrophied arteries have precisely the same structure and appearance as those of normal arteries; but their muscular tissue is increased in amount. This has been acknowledged by all who have taken the trouble to examine the

Fig. 3.—Artery with hypertrophied walls from the subcutaneous tissue, in a case of chronic Bright's disease. The canal of the artery is injected.—X 200.

specimens, including some of the most experienced microscopists and physiologists in London.

What is the explanation of this remarkable hypertrophy of the arterial walls? The theory which led me to search for, and to discover the anatomical facts, probably affords the true explanation of the phenomena.

In consequence of degeneration of the kidney, the blood is morbidly changed. It contains urinary excreta, and it is deficient in some of its own normal constituents. It is, therefore, more or less unsuited to nourish the tissues—more or less noxious to them. The minute arteries throughout the body resist the passage of this abnormal blood. The left ventricle, therefore, makes an increased effort to drive on the blood. The result of this antagonism of forces is, that the muscular walls of the arteries, and those of the left ventricle of the heart, become simultaneously and in an equal degree hypertrophied. The persistent overaction of the muscular tissue, both cardiac and arterial, is found registered after death in a conspicuous and unmistakable hypertrophy.

The hypertrophy of the minute arteries of the kidney is best explained by a reference to the analogous phenomena of apnœa. A ligature on

the trachea of a dog destroys life in a few minutes. The immediate cause of death is the arrest of blood in the lungs. The left cavities of the heart are found after death nearly empty; the right cavities, the pulmonary artery with its branches, and the systemic veins, are much distended; the pulmonary capillaries are nearly bloodless; and the lungs, consequently, collapse to an extreme degree when the chest is opened.

The theory is, that, when the respiratory changes are suspended by the exclusion of air, the minute pulmonary arteries, under the influence of the vaso-motor nerves, by their contraction arrest the flow of blood. Similar phenomena occur during a fit of spasmodic asthma. Bronchial spasm lessens the supply of air; then the contraction of the minute pulmonary arteries, in a corresponding degree, checks the circulation. The skin becomes cold and blue, the pulse small and feeble; and the patient is apparently at death's door. When the bronchial spasm relaxes, the air gets ready access to the pulmonary cells, then the arterial resistance ceases, and respiration and circulation again become free. In like manner, when the secreting tissue of the kidney is partially disorganised, the working power of the gland is lessened, and it requires less blood. The minute renal arteries regulate the blood-supply in accordance with the diminished requirements of the gland. This continues and increases month after month, year after year; and the result of the persistent overaction of the minute renal arteries is, that their muscular walls become hypertrophied. This hypertrophy of the muscular walls of the minute renal arteries occurs in all forms of chronic Bright's disease; in the small, red, granular kidney; and in the large, white, smooth kidney. The hypertrophy of the muscular walls is quite distinct from the degeneration of the arterial tunics which occurs during the progress of that form of disease of the kidney to which the term "lardaceous" is commonly applied.

In conclusion, I may mention some of the phenomena of chronic Bright's disease which are explained by the facts to which I have referred.

1. The hypertrophy of the left ventricle is explained. That which Dr. Bright long ago surmised is now a demonstrated fact. There *is* an impediment to the passage of the altered blood "through the distant subdivisions of the vascular system"; hence arise greater action of the heart, and hypertrophy of its walls.

2. The full, hard, radial pulse, and the increased pressure on the arterial walls indicated by the sphygmograph, are explained by the co-

existence of excessive cardiac force with an equal, or more than equal, excess of arterial resistance.

3. The excessive dryness of the skin, and the difficulty of exciting diaphoresis even by the hot-air bath, are accounted for by overaction of the hypertrophied subcutaneous arteries resisting the relaxing effect of external warmth, which has so powerful an influence upon the healthy skin.

4. One result of excessive resistance throughout the whole systemic arterial system must be to impede the passage of blood from the venous system through the lungs, and thus to favour the occurrence of dropsy by an influence not unlike that of a defective valve on the left side of the heart. The influence of arterial resistance in retarding the circulation will mainly depend upon the extent to which this is counteracted by hypertrophy of the left ventricle of the heart.

5. Lastly, the not infrequent occurrence of cerebral hæmorrhage is explained. While the minute cerebral arteries resist the passage of the blood, the strong left ventricle is forcibly driving it onwards. In the struggle between the two contending forces an artery gives way, and the result is cerebral hæmorrhage and apoplexy.

POSTSCRIPT.—During the discussion which followed the reading of this paper, I was asked for an explanation of the thick inner layer of longitudinal fibres, which forms so conspicuous a feature of the hypertrophied *renal* arteries. (See Fig. 2.) I replied, that I had in vain sought for a satisfactory explanation. It has since occurred to me that the following may be the physiological explanation of the anatomical fact. The renal arteries are of large size, and have a very short course from the aorta into the gland. During the progress of chronic Bright's disease, the circulation through the kidney is much obstructed; so that, with a large afflux of blood to the diseased organ, there is a much impeded outflow. The degrec of impediment to the circulation through the kidney may be estimated by the hypertrophy of the arterial walls. This usually appears to be greater in the renal arteries than in those of any other tissue. I have often seen the thickness of the arterial wall equal to the diameter of the canal of the artery. The tendency of the distending force of the hypertrophied left ventricle obviously is to elongate as well as to dilate the arteries. We have a good illustration of this in cases of incompetent aortic valves with a dilated and hypertrophied ventricle, when the subcutaneous arteries can be seen and felt to swell and lengthen, and so to move beneath the skin, at each systole of the ventricle. That the minute renal arterics, in cases of chronic

Bright's disease, become elongated, is shown by their tortuous course, which is visible in some microscopic specimens. (See Fig. 2.) This elongation of the vessels would be checked by increasing the thickness and strength of the longitudinal fibres; and here probably we have the physiological interpretation of the hypertrophied longitudinal layer. This is more conspicuous in the renal arteries, because those vessels are subjected to greater strain and pressure than the arteries of other tissues, and because in them the general hypertrophy of the walls is excessive.

In confirmation of this view, I may refer to some specimens in my possession from a case of chronic Bright's disease, in which, with only moderate hypertrophy of the circular fibres of the minute renal arteries, the inner longitudinal layer is scarcely visible. These arteries have their canals here and there irregularly dilated, and some of them have a course almost as tortuous as that of the convoluted uriniferous tubes. I infer that in this instance the arteries had become unusually elongated, tortuous, and dilated, in consequence of the absence, or rather the slight degree, of that protective hypertrophy which is so generally present in cases of chronic Bright's disease.

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